ORIGINAL ARTICLES

CARDIAC CARE AFTER DECOMPENSATION*

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DISCUSSION *by George Dock, M. D., Pasadena; William H. Leake, M. D., Los Angeles; Eugene S. Kilgore, M. D., San Francisco.*

THE heart is an organ of great reserves, possibly more so than other organs of the body. Sir Thomas Lewis says that when a heart has broken enough to have the early symptoms of decompensation, such as dyspnea, edema, etc., it has lost nine-tenths of its powers. If we accept this theoretical estimate of its reserves as a practical idea, it is clear to see that, when a decompensated heart is restored just enough to lose these early symptoms, it has gained very little, relatively speaking. The enormous margins that it should have are still lacking. It is surely a great mistake for the physician to feel satisfied with relief of the gross symptoms of decompensation. He should feel it his duty to continue supervision of the case until the margins of the heart can no longer be improved. If he has a true understanding of cardiology, he will know ways and means of assisting his patient to restore the badly needed reserves in some measure at least. On this account, he should hold a steady vigilance over these cases for long periods after the gross symptoms of decompensation have disappeared. It is only in this manner that the best results can be obtained, and the time of the next failure be held off.

PURPOSE OF THIS PAPER

This paper has nothing new to present. Its object is only to reiterate and reëmphasize the points that are so important in cardiac care at the time of this period after decompensation. Some patients stop treatment nicely and go on for some time with good compensation. Others are unable to stop treatment at all, or if they do, go for a short time only.

I would like at this time to emphasize the points that help us to tell ahead of time to which class these patients belong, and also assist us in their treatment after they are properly classified. There are many points that are helpful, but time and space will not permit of their discussion. I would like to take the matter up under the four following broad headings: 1. Cardiac rate. 2. Fibrillation. 3. Hypertension. 4. Venous pressure.

CARDIAC RATE

Counting the pulse is such a simple thing to do that we sometimes fail to think of it in terms of the proper respect. Our forbears in medicine learned much from it—more, I sometimes think, than we who have such an advantage over them in advanced knowledge of anatomy, physiology, biochemistry, etc. At any rate, it is still helpful to know the rate of the heart, and a little more so after decompensation than at other times.

After the withdrawal of digitalis, if the rate is inclined to stay above the conventional 72 while at rest, it is well to be wary and throw protection around that case in the way of restrictions and possibly the continuance of digitalis. The higher it stays the greater and more binding is the need for care and watchfulness. The conscientious physician will not drop his patient simply because dyspnea, edema, cyanosis, enlarged liver, etc., have disappeared, but will continue his supervision until he knows just how the heart is going to carry its load, and something about how much margin it has left after the load is carried.

As the rate of the heart increases, it is increased at the expense of diastole, which is the resting period of the heart. It is this fact that works the mischief. The coronary circulation goes into the heart only during diastole. Therefore, as this period is shortened the nutrition of the heart is also "shortened," and it is less able to do its allotment of work. The Thebesian veins are emptied by the squeeze of systole, and then once more the coronary circulation has its chance to nourish the heart during the relaxation of the next diastole.

The exhaustion of any muscle is due to the collection of lactic acid and other metabolites within it. Cardiac muscle seems to be a little more sensitive to lactic acid than the somatic muscles. When one looks at the facilities provided for the venous drainage of the heart, it is not hard, despite this fact, to understand its marvelous powers. This excellent venous drainage keeps it free from lactic acid concentration and the presence of harmful metabolites, and it is therefore hard to exhaust under normal conditions. Now, if a good, long diastole and free venous drainage are necessary under normal conditions, surely they are much more so under abnormal conditions, where a damaged heart is laboring under great handicaps to carry on its work. Many of these handicaps we are powerless to remove; but this question of rate is somewhat under our control, and we should at least watch it carefully under stressful conditions and use whatever powers we have to assist the crippled heart to maintain a moderately slow rate, get good nutrition, and stay free of metabolite concentration and exhaustion, thus building up as much reserve as is possible.

One of the most beneficial actions of digitalis is its ability to slow the rate of the heart, and this is many times the thing that breaks the vicious cycle of decompensation and starts the heart on its road to recovery. Now, after recovery from the congestive load of decompensation, the heart is often just getting by and its margins very, very small. If under these circumstances the patient is discharged, and has no further care or observation, it can very easily mean the early onset of another decompensation. On the other hand, if proper care is given and when the rate tends to get fast, digitalis is continued, the long diastole is maintained and the patient is protected over a long enough period, the whole picture is changed and the prognosis greatly enhanced.

* Read before the General Medicine Section of the California Medical Association at the sixty-fourth annual session, Yosemite National Park, May 13 to 16, 1935.

In other words, there are patients who are never able to stop digitalis after decompensation, and watching the rate is one of the ways of spotting these cases. The higher the rate the more dangerous it is to stop digitalis; because, as the rate goes up, lactic acid concentration, the oxygen debt, and other important factors in the vicious cycle of decompensation once more establish themselves, and "the fat is in the fire."

FIBRILLATION

The second thing in our list of four major points is *fibrillation.* This occurs most in thyroid and rheumatic hearts, but may also occur in degenerative lesions quite often. From the standpoint of decompensation, it is the most important arrhythmia because of its relative frequence, as well as from its ability to damage the heart. Fibrillation takes the honors in any list of arrhythmias for many reasons.

The rate of the heart is a prime consideration always in dealing with this arrhythmia. The menace of a fast heart rate has already been dealt with, but all that has been said about the rate is doubly binding in fibrillation. A fast rate in this case is not only bad for all the reasons previously named, but for others also. The prognosis of compensation varies directly with the ability to slow the heart. It is well understood by cardiologists that hearts fibrillating on a slow rate do nearly as well as those on a regular sinus rhythm. I have on rare occasions seen these patients hospitalized and put through extensive procedures in an effort to return them to a normal rhythm. In my opinion they had better be left alone.

Pulse Deficit.—In all fibrillating hearts, some of the contractions are weaker than others, and at times these grow so weak as to be unable to lift the aortic leaflets at all, and hence fail to propel the blood stream in its onward direction. It is a very exhausting thing for the heart to have a contraction that accomplishes nothing. It wastes and dissipates its energy in a most harmful manner. As the rate of the fibrillating heart goes up, these weak ineffectual contractions increase very rapidly in number and in proportion to the effectual beats. We have only one method of preventing these contractions, that is to reduce the rate, thereby greatly reducing their number or proportion. As has been explained, a fast rate is exhausting to any heart; but to a fibrillating heart it is doubly so because, in addition to the shortened diastole and its ill effects, is added the terrific loss of energy due to these weak and ineffectual contractions that become so numerous with the increase of rate. Fortunately, we have in digitalis a splendid instrument to assist us in controlling the rate and we must not fail to use it intelligently. We must not hesitate to continue its use indefinitely, if it is necessary to protect a struggling heart from the short diastole in any rhythm and from the pulse deficit of fibrillation.

The pulse deficit is so easily figured, by subtracting the rate as obtained at the wrist from the rate as obtained at the apex, that it pays everyone to do this, and have it charted if the patient is hospitalized, or has sufficient nursing care. It is very striking to watch the graph of the pulse deficit correct itself as the case is digitalized and otherwise compensated by good care. Auricular fibrillation is a vicious complication, and this by no means exhausts the listing of its dangers or the charges that can be brought against it. This only serves to emphasize the two most serious things that we need to watch in a heart that is struggling to regain power and reserves after a failure, when fibrillation is present. To do this is so easy, simple and practical that it needs to be constantly emphasized.

HYPERTENSION AND DEGENERATION

I wish to say that it is not the intention to in any way discuss hypertension. Rather, I wish to emphasize some points of our knowledge of physiology of the circulation and its hydrodynamics that can be used in a practical clinical way in thinking about these hearts that have once decompensated.

If hypertension is present and cannot be controlled, it greatly detracts from the general outlook, as far as preventing recurrences of the decompensation are concerned. So we should hold carefully in mind this fact, and if our patient has a hypertension after decompensation that we cannot reduce we must be cautious, for this case is in direct proportion to the degree and severity of the hypertension.

The reasons, while obvious, will bear repeating. The systolic pressure measures the working force of the heart. If it is high, it means that the heart is maintaining circulation by an expenditure of power that is at a much higher level than otherwise. This obviously will exhaust it more rapidly. Therefore, if it can be brought down by any means whatsoever, it should be done so as to conserve the energy of a struggling heart.

The diastolic pressure is the measure of pressure at the end of ventricular diastole. It measures the peripheral resistance as determined largely by the tonus of the arterioles. It is not so subject to influence nor to variations as the systolic pressure, and on this account is a better indicator of the work required of the heart. If the diastolic pressure is high, it simply means that the heart must expend that much more energy to lift the aortic valves before it can expend any energy in actually propelling the blood along the highway of the arterial system, toward the end of nourishing the body and otherwise carrying on the metabolism of the system or relieving the congestive load. On this account, if the diastolic pressure is high, it greatly adds to the burden of a heart that has been once decompensated and is struggling to carry on with greatly narrowed margins. Every effort should be put forth to reduce it.

The pulse pressure represents the changes in pressure that occur in any given artery as a result of the heart's contractions. This is, therefore, a measure of the force that is driving the blood toward the periphery. This is an accurate way of estimating the "load" that the heart must carry to maintain circulation. When this pulse pressure

is high the heart is carrying more of a load to maintain circulation than it should. It is clear then that to increase the power and reserves of the heart, it is well to reduce this pulse pressure if it is at all possible. Practically speaking, there are five diseases that carry an increased pulse pressure. This little list should be kept well in mind, for whenever you meet one of them in routine examination you at once know that you are dealing with a heart that is expending more energy than it should to do its work. It this happens to be after a decompensation, it is also very evident that it would be well to do what is possible to reduce this pulse pressure toward the end of saving the heart the expenditure of unnecessary energy. The turning of this energy rather into reserves is of prime importance. The list is as follows: hypertension; hyperthyroidism; aortic regurgitation; heart-block; and acromegaly.

However, it is unfortunately necessary to admit that there are many cases of essential hypertension that we are unable to influence.

If there is any means that will hold the pressure down for any case of decompensation, as the patient is getting around again and beginning to look with hope to the future, it should be used. Its successful and continued use may add much time to the productive activity of the individual.

The Degenerative Lesions.—The greater the degree of arteriosclerosis atheromatous disease and fibrotic degeneration, the greater are the dangers to the heart after decompensation. This is one of the reasons why digitalis sometimes gets such a poor response in the aged. Nothing can make fibrotic tissue contract or assist the heart in its contractions. Therefore, when fibrotic replacement exceeds a certain definite amount, the muscle that is remaining finally becomes inadequate. If compensation is restored in such a heart, its ability to hold that compensation is, of course, in direct relation to the degree of degeneration that has occurred in it. It is seldom that a patient exhibiting pulsas alternans lives more than two or three years after its permanent onset under any circumstances, to say nothing of one that has been or is decompensating.

Extensive distortion of the great vessels over the heart, the presence of extensive hypertrophy of the myocardium, and the presence of pulsas alternans should always be taken into consideration when thinking of the prognosis and aftercare of a decompensated heart.

VENOUS PRESSURE

The normal venous pressure lies somewhere between 8 and 20 millimeters of water. In 1915 A. H. Clark published "A Study of the Diagnostic and Prognostic Significance of Venous Pressure in Cardiac Disease." He established at this time the fact that a venous pressure of 20 millimeters of water or over was a sign of cardiac failure. The moment the right ventricle allows the slightest residue of blood after systole, just that moment the venous pressure starts to rise. Eyster and Middleton in their publications have repeatedly pointed out the clinical value of this point. In other words, we should remember that venous pressure is an excellent indicator of right heart efficiency and, generally speaking, of the myocardium as a whole.

It is unfortunate that we do not possess better means of making a clinical, bedside reading of venous pressure than we do. Middleton has produced an instrument for doing it, but it has not met with wide recognition and use. However, there are many practical ways of estimating it clinically, and we need to constantly remind ourselves of these things. One of these things is that urine volume and venous pressure vary inversely. It is, of course, of no trouble to turn this to clinical use.

We need no instrument to enable us to observe the veins of our patient. If they stand out like whipcords it should make us stop and think, and of course means an increased venous pressure. The veins of the hands and arms should collapse or at least reach a theoretical zero of pressure when the arm lies at the cardiac level in a relaxed state. If they do not do so, it means an increased venous pressure. The veins of the head and neck, for obvious reasons, should have a negative pressure in them, more than that of the veins of the lower parts of the body and should be collapsed. If they are not so, then we know there is an increased venous pressure and a right heart that is threatening to decompensate if it has not already started to do so.

These are valuable clinical findings and indicators. When a decompensated case is convalescing and we are in doubt about stopping digitalis or about how much activity we can allow the patient, these are very important things to think about to guide us in forming that opinion. It is interesting to note in passing that Eyster and Middleton have shown venous pressure after venesection to be an excellent indicator of prognosis as to whether or not compensation will be restored after venesection in decompensation. "If the heart muscle responds adequately to the reduced load, the venous pressure will remain low, and the subjective and objective evidence of cardiac decompensation will subside. On the other hand, an early sharp rise in the venous pressure, or a steady return to its original high level, points to a lack of myocardial reserve and no continued clinical improvement can be expected."

TO SUMMARIZE

To review, in closing, our discussion of this clinical question of the care of the heart after decompensation, I wish to emphasize that I believe they fail of receiving the proper consideration during this time. I believe these patients could live much longer and with greater comfort and efficiency if more attention were given them after the gross signs of decompensation have disappeared. This paper is put forward as a means of emphasizing important landmarks that should guide us in our decision regarding them.

If digitalis needs to be continued to protect the weakened reserves of the heart, let us not

hesitate to use it—indefinitely, if need be. Restrictions regarding rest and all activities should be continued in proportion to the need.

The four major things to be considered in deciding the need for continuation of treatment are as follows:

1. If the rate is above 72, at complete rest.

2. If fibrillation is present with a large pulse deficit (the faster the rate the more binding this statement).

3. If venous pressure is increased, as evidenced by prominent veins, especially in head and neck, or the arm if fully relaxed and resting at the cardiac level.

4. If hypertension is present, with markedly increased pulse pressure.

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DISCUSSION

GEORGE DOCK, M. D. (94 North Madison Avenue, Pasadena).—As I followed Doctor Clarke's presentation, I realized that I was listening to an unusually complete and sensible treatise on a very important class of patients. I noticed nothing important to criticize, but thought that here was a good paper to study when it appeared in print. It would be useful to every reader, no matter what his line of practice, to read paragraph by paragraph and ask himself whether he has known and observed all the points presented. Let me mention some of the points that I think especially worthy of emphasis. The matter of continuing supervision until the very best results have been obtained, is still occasionally forgotten. The matter of pulse counting is just as important, and it is strange that while instruments of precision have increased the value of the pulse in diagnosis, not a few patients express surprise when they find their radials objects of interest, and tell us they understood pulse feeling is no longer necessary. I am pleased to see the assumption that digitalis is the great remedy in patients with decompensated hearts. The physician should be very sure he has given digitalis a chance before he discards it for something else. The discussion of pulse deficit is very good, and here, too, minute observation of the situation is not sufficiently carried out. The pulse count is noted without regard to the apex beat only too often. At one time Sir James Mackenzie would not allow pulse counts made by nurses: all had to be made by physicians and checked by polygraph. Of course the latter can be dispensed with, but never combined counting of heart and radial. I close by repeating my advice as to careful study of the paper.

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WILLIAM H. LEAKE, M. D. (1930 Wilshire Boulevard, Los Angeles).—The after-care of patients who have recovered from congestive heart failure is of vital importance. It is regrettable that cardiac patients are discharged frequently by the physician with little or no advice concerning subsequent activities and medication. Systematic follow-up examinations are of great value in detecting signs of early heart failure, thereby enabling the physician to institute appropriate treatment.

Digitalis is a drug of inestimable value in the treatment of congestive heart failure but, unfortunately, it is used too sparingly by many general practitioners. The lack of response to digitalis in congestive heart failure accompanying the rheumatic and the degenerative types of heart disease, is frequently the result of inadequate dosage. This is especially true of the first attack. It must be remembered that with each "break" in compensation, response to treatment is less satisfactory. Heart failure secondary to syphilitic heart disease is notoriously resistant to digitalis therapy.

The necessity for counting the heart rate at the apex when arrhythmia is present must be emphasized. It is my opinion that the apex rate should be taken routinely in all cardiac patients, regardless of the rhythm. The frequent presence of "pulse deficit" in association with untreated auricular fibrillation, is well recognized. Attention should be called, however, to the existence of "pulse deficit" in certain cases of extrasystolic arrhythmia, auricular flutter and paroxysmal ventricular tachycardia. The "pulse deficit" disappears, as a rule, when the heart rate falls below 80 per minute. Attempts to revert established auricular fibrillation to normal rhythm by the use of digitalis are usually unsuccessful. This is of no importance, inasmuch as this type of irregularity causes little discomfort, provided the apex rate is not too rapid. The dosage of digitalis in chronic cardiac patients should be sufficient to maintain the heart rate between 50 and 70 per minute when the individual is at rest.

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EUGENE S. KILGORE, M. D. (490 Post Street, San Francisco).—Doctor Clarke rightly emphasizes the importance of the less obvious in the care of heart patients; and what he says might well be applied also to the period before definite decompensation has occurred. The earlier and milder manifestations of disease should receive more attention in medical teaching and in medical practice, and this is especially true of heart disease.

In the particular problem under discussion—the gauging of the return of cardiac reserve after decompensation—my interpretation of the criteria suggested by Doctor Clarke differs in certain respects from his. The pulse rate is affected by many factors other than the degree of cardiac reserve present. Many persons obviously in good health have, under conditions of rest, a pulse rate much over the conventional seventy-two. I once observed a young man who undoubtedly had excellent cardiac reserve (he was a very good sprinter), but whose pulse rate at rest was 180 to 200. Change in pulse rate may be of greater significance than the rate itself. A declining pulse rate is, of course, the rule during recovery from congestive failure; but while this is an important index of improvement, it does not seem to me to be the best general guide in controlling digitalis dosage, for the reason that the chief effect of the drug is not reflected in the rate of the sinus pacemaker. In treating congestive failure, the best effect of digitalis appears just below the toxic level; and it is usually desirable, after digitalis saturation, to continue the drug at a level a little below this and well through the period of convalescence, the dosage being guided by the appearance of toxic manifestations on the one hand and, on the other, by the ensemble of favorable effect on the pulse, the respiration, kidney function, the color and general appearance of the patient. Patients who have atrial fibrillation form the one notable exception where usually the pulse rate alone is an all-sufficient indicator of the digitalis effect.

The rising blood pressure frequently seen during recovery from decompensation is usually a return toward a higher level which antedated the failure. With Doctor Clarke, I see in this the disadvantage of added work for the heart, but it may also mean a better flow through the coronaries and renal vessels; so that, while unfortunate in certain ways, in general it is to be regarded as a favorable omen. A similar interpretation is applicable to the expanding pulse pressure frequently seen during recovery from heart failure.

To the items which Doctor Clarke has mentioned as guides in the management of convalescence, I would add the general appearance of the patient (facies, respiration, color, kidney function, etc.), and the patient's own observations. Remembering that, before any important cardiac reserve is established, the patient may become entirely comfortable at rest, the return to activity must, of course, be very gradual. For patients who have been orthopneic, the return to a horizontal sleeping posture should be accomplished cautiously. This and the later getting out of bed, walking a few steps, climbing stairs, etc., should all be regulated, not merely within the limits of comfort, but within limits which produce very little effect on pulse rate and respiration.

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DOCTOR CLARKE (Closing).—We have all observed hearts with a high rate that were slowed by exercise. It is quite probable that, could Doctor Kilgore have counted the pulse of his sprinter when he was in the middle of the hundred-yard dash, he would have found his rate very much lower than 180 or 200. The difficulty with this suggestion of

Doctor Kilgore's is that the sprinter's heart was undoubtedly of a sinus rhythm. Had he been working under an arrhythmia, I am sure he would not have gotten very far on the cinder pathway without going into a heap. The faster a fibrillating heart goes the greater is its arrhythmia and the more damage to its reserves. Surely, Doctor Kilgore would not try to tell us that the slowing of a pulse rate and the lengthening of its diastolic period is not good treatment, and an important point to observe in recovery from decompensation.

THE ASCHHEIM-ZONDEK TEST FOR PREGNANCY*

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DISCUSSION *by Gertrude Moore, M. D., Oakland; Alvin G. Foord, M. D., Pasadena; George D. Maner, M. D., Los Angeles.*

THE early work of Aschheim and Zondek[1] on sex glands and sex hormones progressed slowly because the rabbit was the only known suitable animal for the purpose. Rabbits became scarce and expensive, and some other animal had to be found. They soon found that immature white mice and immature white rats were suitable.

Working with immature female white mice they[2] showed that ovarian hormone in the blood or in the urine of a woman does not indicate pregnancy, and that fairly large quantities of ovarian hormone can be demonstrated in the blood and in the urine of women in many conditions besides pregnancy. They found that presence in the blood and in the urine of a substance which in the test animal causes hemorrhagic follicles and cellular proliferation in the ovaries, and probably premature ovulation like the reaction following the injection of an extract of the anterior portion of the pituitary gland, indicates pregnancy. They considered this substance to be a hormone secreted by the anterior portion of the pituitary gland.

VALUE OF STUDIES OF ASCHHEIM AND ZONDEK

Aschheim and Zondek[3] then showed that the test is practically specific for pregnancy by applying it to many other conditions with negative results. Twenty-six urines of normal, menstruating women all gave negative results. Six urines from old women gave negative results. Sixteen urines of unknown source gave results that agreed with the clinical findings. Three urines from women with irregular menstruation gave negative results. Of sixteen urines from men, one gave a positive result which could not be explained. Fifteen urines from women with internal diseases gave negative results. One urine from a woman with cystitis gave a positive result. Of three urines from acromegaly, two were highly toxic; they killed all the animals and one gave a positive result. Thyroid diseases gave negative results. Of twelve urines from women with infections of the genital organs, ten gave negative results and two gave positive results, and one of these had received Roentgen-ray treatment. Ten benign ovarian tumors and eighteen uterine myomas gave negative results. One case of uterine myoma gave a positive result and was found to be pregnant. Of sixty urines from women with cancer, fifty-eight gave negative results and two gave positive results. The two urines that gave positive results came from women with carcinoma of the ovary. Of 197 urines from normal pregnant women, 195 gave positive results and two gave negative results, and these gave positive results a few days later. After delivery, the results were always negative in eight days and frequently in five. After death of the fetus in utero, and after abortion the results became negative as soon as after normal delivery. In tubal pregnancy the results were positive while the fetus was alive, but soon became negative after the death of the fetus. Two urines from women with hydatidiform mole gave positive results.

In 1929, Friedman[4] reported on thirty-six urines from pregnant women, all of which gave positive results in rabbits.

OUTLINE OF AUTHOR'S STUDIES

The work reported here was done on rabbits, and the following questions are considered:

1. The most suitable age of the rabbit.
2. The time required for the reaction in the rabbit.
3. The Aschheim-Zondek test with urine from normal men and with urine from normal nonpregnant women.
4. The Aschheim-Zondek test in pregnancy.
5. Preservation of urine for the Aschheim-Zondek test.
6. The Aschheim-Zondek test in various pathologic conditions.

The Age of the Rabbit.—Female rabbits two months old, from three to four months old, and some adults were injected with urine from the same pregnant woman. The rabbits between three and four months old reacted better than the younger or older rabbits. I prefer rabbits between three and four months old.

The Reaction in the Rabbit.—A number of rabbits were inoculated at about the same time with urine from a pregnant woman; then some were examined after twenty-four hours, some after forty-eight hours, some after seventy-two hours, some after five days, some after seven days, some after ten days and some after twenty-one days. Twenty-four hours after injection there was a slight reaction in the ovaries; it was good after forty-eight hours; it reached its height in four or five days, then gradually subsided, leaving scarred masses with a few hemorrhagic points by the end of twenty-one days.

Urines from Normal Persons.—Five urines from normal men, five from normal nonmenstruating women, and five urines from five menstruating women all gave negative results.

Urines from Pregnant Women.—All rabbits were between three and four months old, and for diagnostic test each rabbit received about 12 cubic centimeters of urine, with a specific gravity of

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